

Ref #	Hits	Search Query	DBs	Default Operator	Plurals	Time Stamp
L1	14	N2 near2 vh nearvk near (gene adj1 III)	US-PGPUB; USPAT; EPO; DERWENT	OR	OFF	2005/06/28 14:12
L2	0	N2 near2 domain near5 binding near5 anchoring	US-PGPUB; USPAT; EPO; DERWENT	OR	OFF	2005/06/28 14:12
L3	0	N2 near2 domain near5 binding near10 anchor	US-PGPUB; USPAT; EPO; DERWENT	OR	OFF	2005/06/28 14:12
L4	0	N2 near5 domain near8 binding near10 anchoring	US-PGPUB; USPAT; EPO; DERWENT	OR	OFF	2005/06/28 14:14
L5	0	N2 near5 Cd80 near5 vh near3 vl	US-PGPUB; USPAT; EPO; DERWENT	OR	OFF	2005/06/28 14:14
L6	0	N2 near5 Cd80 near5 vh	US-PGPUB; USPAT; EPO; DERWENT	OR	OFF	2005/06/28 15:02
L7	7	yeast near10 anchor?	US-PGPUB; USPAT; EPO; DERWENT	OR	OFF	2005/06/28 15:15
L8	1	("5837242").PN.	USPAT; EPO	OR	OFF	2005/06/28 16:36
L9	1	("5869620").PN.	USPAT; EPO	OR	OFF	2005/06/28 15:54
L10	11	CD80 same CD3 same CD16	USPAT; EPO; DERWENT	OR	OFF	2005/06/28 16:43
L11	111	N2 same (vh or vl)	USPAT; EPO; DERWENT	OR	OFF	2005/06/28 16:43
L12	1	N2 same (vh or vl) same domain	USPAT; EPO; DERWENT	OR	OFF	2005/06/28 16:46
L13	39	N2 near8 (vh or vl)	USPAT; EPO; DERWENT	OR	OFF	2005/06/28 16:46
L14	32	N2 near5 (vh or vl)	USPAT; EPO; DERWENT	OR	OFF	2005/06/28 16:46

L15	0	N2 near5 (vh or vl) near8 (gene adj1 III)	USPAT; EPO; DERWENT	OR	OFF	2005/06/28 16:56
L16	0	N2 near5 (vh or vl) near15 III	USPAT; EPO; DERWENT	OR	OFF	2005/06/28 16:47
L17	0	N2 near5 (vH or vL) near15 III	USPAT; EPO; DERWENT	OR	OFF	2005/06/28 16:48
L18	0	N2 near5 (vH or vL) near15 gene	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/06/28 16:48
L19	0	N2 near10 (vH or vL) near20 gene	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/06/28 16:50
L20	1	("6027930").PN.	USPAT; EPO	OR	OFF	2005/06/28 16:50
L21	0	N2 near8 (gene adj1 III)	USPAT; EPO; DERWENT	OR	OFF	2005/06/28 16:56
L22	0	N2 near20 (gene adj1 III)	US-PGPUB; USPAT; EPO; DERWENT	OR	OFF	2005/06/28 16:57
L23	1	N2 near20 phage near20 (vh or vl or antibody)	US-PGPUB; USPAT; EPO; DERWENT	OR	OFF	2005/06/28 16:57

L15	0	N2 near5 (vh or vl) near8 (gene adj1 III)	USPAT; EPO; DERWENT	OR	OFF	2005/06/28 16:56
L16	0	N2 near5 (vh or vl) near15 III	USPAT; EPO; DERWENT	OR	OFF	2005/06/28 16:47
L17	0	N2 near5 (vH or vL) near15 III	USPAT; EPO; DERWENT	OR	OFF	2005/06/28 16:48
L18	0	N2 near5 (vH or vL) near15 gene	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/06/28 16:48
L19	0	N2 near10 (vH or vL) near20 gene	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/06/28 16:50
L20	1	("6027930").PN.	USPAT; EPO	OR	OFF	2005/06/28 16:50
L21	0	N2 near8 (gene adj1 III)	USPAT; EPO; DERWENT	OR	OFF	2005/06/28 16:56
L22	0	N2 near20 (gene adj1 III)	US-PGPUB; USPAT; EPO; DERWENT	OR	OFF	2005/06/28 16:57
L23	1	N2 near20 phage near20 (vh or vl or antibody)	US-PGPUB; USPAT; EPO; DERWENT	OR	OFF	2005/06/28 17:44
L24	1	("6667150").PN.	USPAT; EPO	OR	OFF	2005/06/28 17:46
L25	14	N2 same coli same domain	US-PGPUB; USPAT; EPO; DERWENT	OR	OFF	2005/06/28 17:46

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NEWS	17	MAY 23	GBFULL enhanced with patent drawing images
NEWS	18	MAY 23	REGISTRY has been enhanced with source information from CHEMCATS
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NEWS	22	JUN 13	FRFULL enhanced with patent drawing images
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=> eukaryotic(8A) (anchoring or anchor or anchored)  
THIS COMMAND NOT AVAILABLE IN THE CURRENT FILE  
Some commands only work in certain files. For example, the EXPAND command can only be used to look at the index in a file which has an index. Enter "HELP COMMANDS" at an arrow prompt (=>) for a list of commands which can be used in this file.

=> file .meeting  
'EVENTLINE' IS NOT A VALID FILE NAME  
Enter "HELP FILE NAMES" at an arrow prompt (=>) for a list of files that are available. If you have requested multiple files, you can specify a corrected file name or you can enter "IGNORE" to continue accessing the remaining file names entered.  
ENTER A FILE NAME OR (IGNORE): ignore  
COST IN U.S. DOLLARS

	SINCE FILE	TOTAL
	ENTRY	SESSION
FULL ESTIMATED COST	0.42	0.42

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=> eukaryotic(8A) (anchoring or anchor or anchored)

L1	4 FILE AGRICOLA
L2	51 FILE BIOTECHNO
L3	0 FILE CONFSCI
L4	0 FILE HEALSAFE
L5	0 FILE IMSDRUGCONF
L6	39 FILE LIFESCI

L7 0 FILE MEDICONF  
L8 16 FILE PASCAL

TOTAL FOR ALL FILES

L9 110 EUKARYOTIC(8A) (ANCHORING OR ANCHOR OR ANCHORED)

=> eukaryotic(8A) (anchoring)

L10 1 FILE AGRICOLA  
L11 9 FILE BIOTECHNO  
L12 0 FILE CONFSCI  
L13 0 FILE HEALSAFE  
L14 0 FILE IMSDRUGCONF  
L15 6 FILE LIFESCI  
L16 0 FILE MEDICONF  
L17 2 FILE PASCAL

TOTAL FOR ALL FILES

L18 18 EUKARYOTIC(8A) (ANCHORING)

=> dup rem

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L19 12 DUP REM L18 (6 DUPLICATES REMOVED)

=> d l19 ibib abs total

L19 ANSWER 1 OF 12 LIFESCI COPYRIGHT 2005 CSA on STN

ACCESSION NUMBER: 2004:40293 LIFESCI

TITLE: Chromosome segregation in Eubacteria

AUTHOR: Pogliano, K.; Pogliano, J.; Becker, E.

CORPORATE SOURCE: Division of Biological Sciences, 9500 Gilman Drive,  
University of California, San Diego, La Jolla, CA  
92093-0349, USA; E-mail: kpogliano@ucsd.edu

SOURCE: Current Opinion in Microbiology [Curr. Opin. Microbiol.],  
(20031200) vol. 6, no. 6, pp. 586-593.  
ISSN: 1369-5274.

DOCUMENT TYPE: Journal

TREATMENT CODE: General Review

FILE SEGMENT: J

LANGUAGE: English

SUMMARY LANGUAGE: English

AB It is now clear that bacterial chromosomes rapidly separate in a manner independent of cell elongation, suggesting the existence of a mitotic apparatus in bacteria. Recent studies of bacterial cells reveal filamentous structures similar to the **eukaryotic** cytoskeleton, proteins that mediate polar chromosome **anchoring** during *Bacillus subtilis* sporulation, and SMC interacting proteins that are involved in chromosome condensation. A picture is thereby developing of how bacterial chromosomes are organized within the cell, how they are separated following duplication, and how these processes are coordinated with the cell cycle.

L19 ANSWER 2 OF 12 BIOTECHNO COPYRIGHT 2005 Elsevier Science B.V. on STN  
DUPLICATE

ACCESSION NUMBER: 2002:35365609 BIOTECHNO

TITLE: The glycosylphosphatidylinositol (GPI) signal sequence of human placental alkaline phosphatase is not recognized by human Gpi8p in the context of the yeast GPI anchoring machinery

AUTHOR: Meyer U.; Fraering P.; Bosson R.; Imhof I.; Benghezal M.; Vionnet C.; Conzelmann A.

CORPORATE SOURCE: A. Conzelmann, Institute of Biochemistry, University of Fribourg, Fribourg, Switzerland.  
E-mail: andreas.conzelmann@unifr.ch

SOURCE: Molecular Microbiology, (2002), 46/3 (745-748), 6  
reference(s)  
CODEN: MOMIEE ISSN: 0950-382X

DOCUMENT TYPE: Journal; Article

COUNTRY: United Kingdom

LANGUAGE: English

SUMMARY LANGUAGE: English

AN 2002:35365609 BIOTECHNO

AB Biosynthesis of glycosylphosphatidylinositol (GPI)-anchored proteins involves the action of a GPI trans-amidase, which replaces the C-terminal GPI signal sequence (GPI-SS) of the primary translation product with a preformed GPI lipid. The transamidation depends on a complex of four proteins, Gaalp, Gpi8p, Gpi16p and Gpi17p. Although the GPI **anchoring** pathway is conserved throughout the **eukaryotic** kingdom, it has been reported recently that the GPI-SS of human placental alkaline phosphatase (hPLAP) is not recognized by the yeast transamidase, but is recognized in yeast that contain the human Gpi8p homologue. This finding suggests that Gpi8p is intimately involved in the recognition of GPI precursor proteins and may also be responsible for the subtle taxon-specific differences in transamidase specificity that sometimes prevent the efficient GPI anchoring of heterologously expressed GPI proteins. Here, we confirm that the GPI signal sequence of hPLAP is indeed not recognized by the yeast GPI-anchoring machinery. However, in our hands, GPI attachment cannot be restored by the co-expression of human Gpi8p in yeast cells under any circumstances.

L19 ANSWER 3 OF 12 BIOTECHNO COPYRIGHT 2005 Elsevier Science B.V. on STN  
DUPLICATE

ACCESSION NUMBER: 2001:32165031 BIOTECHNO

TITLE: Comparative analysis of the nonA region in Drosophila identifies a highly diverged 5' gene that may constrain nonA promoter evolution

AUTHOR: Campesan S.; Chalmers D.; Sandrelli F.; Megighian A.; Peixoto A.A.; Costa R.; Kyriacou C.P.

CORPORATE SOURCE: C.P. Kyriacou, Department of Genetics, University of Leicester, Leicester LE1 7RH, United Kingdom.  
E-mail: cpk@leicester.ac.uk

SOURCE: Genetics, (2001), 157/2 (751-764), 52 reference(s)  
CODEN: GENTAE ISSN: 0016-6731

DOCUMENT TYPE: Journal; Article

COUNTRY: United States

LANGUAGE: English

SUMMARY LANGUAGE: English

AN 2001:32165031 BIOTECHNO

AB A genomic fragment from Drosophila virilis that contained all the no-on-transientA (nonA) coding information, plus several kilobases of upstream material, was identified. Comparisons of nonA sequences and the gene nonA-like in D. melanogaster, a processed duplication of nonA, suggest that it arose before the split between D. melanogaster and D. virilis. In both species, another gene that lies <350 bp upstream from the nonA transcription starts, and that probably corresponds to the lethal gene l(1)i19, was identified. This gene encodes a protein that shows similarities to GPI1, which is required for the biosynthesis of glycosylphosphatidylinositol (GPI), a component for **anchoring** **eukaryotic** proteins to membranes, and so we have named it dGpi1. The molecular evolution of nonA and dGpi1 sequences show remarkable differences, with the latter revealing a level of amino acid divergence that is as high as that of transformer and with extremely low levels of codon bias. Nevertheless, in D. melanogaster hosts, the D. virilis fragment rescues the lethality associated with a mutation of l(1)i19e, as

well as the viability and visual defects produced by deletion of nonA.sup.-. The presence of dGpi1 sequences so close to nonA appears to have constrained the evolution of the nonA promoter.

L19 ANSWER 4 OF 12 BIOTECHNO COPYRIGHT 2005 Elsevier Science B.V. on STN  
ACCESSION NUMBER: 2000:30616635 BIOTECHNO  
TITLE: Cloning of murine glycosyl phosphatidylinositol anchor attachment protein, GPAA1  
AUTHOR: Hiroi Y.; Chen R.; Sawa H.; Hosoda T.; Kudoh S.; Kobayashi Y.; Aburatani H.; Nagashima K.; Nagai R.; Yazaki Y.; Medof M.E.; Komuro I.  
CORPORATE SOURCE: I. Komuro, Dept. of Cardiovascular Medicine, Univ. of Tokyo, Graduate School of Medicine, 7-3-1 Hongo, Bunkyo-ku, Tokyo 113-8655, Japan.  
E-mail: komuro-tyky@umin.ac.jp  
SOURCE: American Journal of Physiology - Cell Physiology, (2000), 279/1 48-1 (C205-C212), 37 reference(s)  
CODEN: AJPCDD ISSN: 0363-6143  
DOCUMENT TYPE: Journal; Article  
COUNTRY: United States  
LANGUAGE: English  
SUMMARY LANGUAGE: English

AN 2000:30616635 BIOTECHNO  
AB Glycosyl phosphatidylinositols (GPIs) are used to anchor many proteins to the cell surface membrane and are utilized in all **eukaryotic** cells. GPI **anchoring** units are attached to proteins via a transamidase reaction mediated by a GPI transamidase complex. We isolated one of the components of this complex, mGPAA1 (murine GPI anchor attachment), by the signal sequence trap method, mGPAA1 cDNA is about 2 kb in length and encodes a putative 621 amino acid protein. The mGPAA1 gene has 12 small exons and 11 small introns, mGPAA1 mRNA is ubiquitously expressed in mammalian cells, and in situ hybridization analysis revealed that it is abundant in the choroid plexus, skeletal muscle, osteoblasts of rib, and occipital bone in mouse embryos. Its expression levels and transamidation efficiency decreased with differentiation of embryonic stem cells. The 3T3 cell lines expressing antisense mGPAA1 failed to express GPI-anchored proteins on the cell surface membrane.

L19 ANSWER 5 OF 12 LIFESCI COPYRIGHT 2005 CSA on STN  
ACCESSION NUMBER: 2001:3362 LIFESCI  
TITLE: Process for immobilizing enzymes to the cell wall of a microbial cell by producing a fusion protein  
AUTHOR: Klis, F.; Schreuder, M.; Toschka, H.; Verrips, C.  
CORPORATE SOURCE: Unilever Patent Holdings B.V.  
SOURCE: (20000222) . US Patent: 6027910; US CLASS: 435/41; 435/69.7; 435/69.8; 435/69.9; 435/172.1; 435/172.3; 435/252.3; 435/320.1.  
DOCUMENT TYPE: Patent  
FILE SEGMENT: W2  
LANGUAGE: English  
SUMMARY LANGUAGE: English  
AB A method is provided for immobilizing an enzyme, comprising immobilizing the enzyme or a functional part thereof to the cell wall of a microbial cell using recombinant DNA techniques. The enzyme is immobilized by linking it to the C-terminal part of a protein that ensures anchoring in the cell wall. Also provided is a recombinant polynucleotide comprising a structural gene encoding an enzyme protein, a part of a gene encoding the C-terminal part of a protein capable of **anchoring** in a **eukaryotic** or prokaryotic cell wall, as well as a signal sequence, in addition to a chimeric protein encoded by the recombinant polynucleotide and a vector and a microorganism containing the polynucleotide. The microorganism is suitable for carrying out enzymatic processes on an industrial scale.



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ACCESSION NUMBER: 2001-0083864 PASCAL  
COPYRIGHT NOTICE: Copyright .COPYRGT. 2001 INIST-CNRS. All rights reserved.  
TITLE (IN ENGLISH): Cloning of murine glycosyl phosphatidylinositol anchor attachment protein, GPAA1  
AUTHOR: HIROI Y.; CHEN R.; SAWA H.; HOSODA T.; KUDOH S.; KOBAYASHI Y.; ABURATANI H.; NAGASHIMA K.; NAGAI R.; YAZAKI Y.; MEDOF M. E.; KOMURO I.  
CORPORATE SOURCE: Department of Cardiovascular Medicine, University of Tokyo Graduate School of Medicine, Tokyo 113-8655, Japan; Department of Pathology, University of Hokkaido School of Medicine, Sapporo, Japan; Institute of Pathology, Case Western Reserve University, Cleveland, Ohio, United States  
SOURCE: American journal of physiology. Cell physiology, (2000), 48(1), C205-C212, 37 refs.  
ISSN: 0363-6143 CODEN: AJPCDD  
DOCUMENT TYPE: Journal  
BIBLIOGRAPHIC LEVEL: Analytic  
COUNTRY: United States  
LANGUAGE: English  
AVAILABILITY: INIST-670B, 354000090369560220

AN 2001-0083864 PASCAL  
CP Copyright .COPYRGT. 2001 INIST-CNRS. All rights reserved.  
AB Glycosyl phosphatidylinositols (GPIs) are used to anchor many proteins to the cell surface membrane and are utilized in all **eukaryotic** cells. GPI **anchoring** units are attached to proteins via a transamidase reaction mediated by a GPI transamidase complex. We isolated one of the components of this complex, mGPAA1 (murine GPI anchor attachment), by the signal sequence trap method, mGPAA1 cDNA is about 2 kb in length and encodes a putative 621 amino acid protein. The mGPAA1 gene has 12 small exons and 11 small introns. mGPAA1 mRNA is ubiquitously expressed in mammalian cells, and in situ hybridization analysis revealed that it is abundant in the choroid plexus, skeletal muscle, osteoblasts of rib, and occipital bone in mouse embryos. Its expression levels and transamidation efficiency decreased with differentiation of embryonic stem cells. The 3T3 cell lines expressing antisense mGPAA1 failed to express GPI-anchored proteins on the cell surface membrane.

L19 ANSWER 7 OF 12 BIOTECHNO COPYRIGHT 2005 Elsevier Science B.V. on STN  
ACCESSION NUMBER: 1998:29012126 BIOTECHNO  
TITLE: Sequence properties of GPI-anchored proteins near the  $\omega$ -site: Constraints for the polypeptide binding site of the putative transamidase  
AUTHOR: Eisenhaber B.; Bork P.; Eisenhaber F.  
CORPORATE SOURCE: B. Eisenhaber, European Molecular Biology Lab., Meyerhofstrasse 1, D-69012 Heidelberg, Germany.  
E-mail: birgit.eisenhaber@embl-heidelberg.de  
SOURCE: Protein Engineering, (1998), 11/12 (1155-1161), 31 reference(s)  
CODEN: PRENE0 ISSN: 0269-2139  
DOCUMENT TYPE: Journal; Article  
COUNTRY: United Kingdom  
LANGUAGE: English  
SUMMARY LANGUAGE: English

AN 1998:29012126 BIOTECHNO  
AB Glycosylphosphatidylinositol (GPI) **anchoring** is a common post-translational modification of extracellular **eukaryotic** proteins. Attachment of the GPI moiety to the carboxyl terminus to-site) of the polypeptide occurs after proteolytic cleavage of a C-terminal

propeptide. In this work, the sequence pattern for GPI-modification was analyzed in terms of physical amino acid properties based on a database analysis of annotated proprotein sequences. In addition to a refinement of previously described sequence signals, we report conserved sequence properties in the regions  $\omega - 11 \dots \omega - 1$  and  $\omega + 4 \dots \omega + 5$ . We present statistical evidence for volume-compensating residue exchanges with respect to the positions  $\omega - 1 \dots \omega + 2$ . Differences between protozoan and metazoan GPI-modification motifs consist mainly in variations of preferences to amino acid types at the positions near the  $\omega$ -site and in the overall motif length. The variations of polypeptide substrates are exploited to suggest a model of the polypeptide binding site of the putative transamidase, the enzyme catalyzing the GPI-modification. The volume of the active site cleft accommodating the four residues  $\omega - 1 \dots \omega + 2$  appears to be .sim. 540 Å<sup>sup</sup>.3.

L19 ANSWER 8 OF 12 BIOTECHNO COPYRIGHT 2005 Elsevier Science B.V. on STN  
 ACCESSION NUMBER: 1995:25214135 BIOTECHNO  
 TITLE: How glycosylphosphatidylinositol-anchored membrane proteins are made  
 AUTHOR: Udenfriend S.; Kodukula K.  
 CORPORATE SOURCE: Roche Institute of Molecular Biology, Roche Research Center, Nutley, NJ 07110, United States.  
 SOURCE: Annual Review of Biochemistry, (1995), 64/- (563-591)  
 CODEN: ARBOAW ISSN: 0066-4154  
 DOCUMENT TYPE: Journal; General Review  
 COUNTRY: United States  
 LANGUAGE: English  
 SUMMARY LANGUAGE: English

AN 1995:25214135 BIOTECHNO  
 AB Glycosylphosphatidylinositol (GPI) linkage is a fairly common means of **anchoring** membrane proteins to **eukaryotic** cells, although the exact function of the GPI linkage is not clear. The nascent form of a typical GPI protein contains a hydrophobic NH.sub.2-terminal signal peptide that directs it to the ER. There the signal peptide is removed by NH.sub.2-terminal signal peptidase. Nascent forms of GPI-linked proteins contain a second hydrophobic peptide at their COOH terminus. The COOH-terminal peptide is also removed during processing and the GPI moiety is ultimately linked to what had been an internal sequence in the nascent protein. Two independent pathways are involved in the biosynthesis of GPI proteins, GPI formation, and processing of the nascent protein with attachment of the GPI moiety. Studies in whole cells and in cell-free systems indicate that structural requirements around the COOH-terminal cleavage site of nascent proteins are similar to those at the cleavage site of NH.sub.2-terminal signal peptidase. However, COOH-terminal processing requires a transamidase for which evidence is presented as well as a proposed mechanism of its action.

L19 ANSWER 9 OF 12 BIOTECHNO COPYRIGHT 2005 Elsevier Science B.V. on STN  
 DUPLICATE  
 ACCESSION NUMBER: 1995:25269437 BIOTECHNO  
 TITLE: Antisense effects of cholesterol-oligodeoxynucleotide conjugates associated with poly(alkylcyanoacrylate) nanoparticles  
 AUTHOR: Godard G.; Boutorine A.S.; Saison-Behmoaras E.; Helene C.  
 CORPORATE SOURCE: Genta, 163 Avenue de Luminy, F-13288 Marseille Cedex 09, France.  
 SOURCE: European Journal of Biochemistry, (1995), 232/2 (404-410)  
 CODEN: EJBCAI ISSN: 0014-2956  
 DOCUMENT TYPE: Journal; Article  
 COUNTRY: Germany, Federal Republic of

LANGUAGE: English

SUMMARY LANGUAGE: English

AN 1995:25269437 BIOTECHNO

AB Oligonucleotides covalently attached to a cholesteryl moiety are more stable in biological media and better taken up by **eukaryotic** cells. However, their **anchoring** in hydrophobic cellular membranes and endosomes after endocytosis restricts their access to cellular nucleic acids. New methods of cellular delivery and the biological activity of the conjugates were studied. The cholesteryl residue was conjugated via disulfide bond to the 5' or 3' terminal phosphate group of two oligodeoxyribonucleotide dodecamers complementary to the mutated region of Ha-ras oncogene mRNA. The conjugates were able to form complementary duplexes with the mutated 27-b target fragment of mRNA but not with the wild-type sequence. Efficient sequence-specific RNase H cleavage of complementary mRNA was induced with low ( $\leq 500$  nM) concentrations of the conjugates. At higher concentrations, this cleavage was progressively inhibited, probably due to an interaction between RNase H and the cholesterol residue. The hydrophobic conjugates could be adsorbed onto poly(isohexylcyanoacrylate) nanoparticles via their cholesteryl moieties and delivered to eukaryotic cells. Cholesterol-conjugated oligonucleotides were able to sequence specifically inhibit the proliferation of T24 human bladder carcinoma cells in culture.

L19 ANSWER 10 OF 12 AGRICOLA Compiled and distributed by the National Agricultural Library of the Department of Agriculture of the United States of America. It contains copyrighted materials. All rights reserved.  
(2005) on STN DUPLICATE 4

ACCESSION NUMBER: 95:14811 AGRICOLA

DOCUMENT NUMBER: IND20446767

TITLE: Intracellular Agrobacterium can transfer DNA to the cell nucleus of the host plant.

AUTHOR(S): Escudero, J.; Neuhaus, G.; Hohn, B.

CORPORATE SOURCE: Friedrich Miescher Institut, Basel, Switzerland

AVAILABILITY: DNAL (500 N21P)

SOURCE: Proceedings of the National Academy of Sciences of the United States of America, Jan 3, 1995. Vol. 92, No. 1. p. 230-234

Publisher: Washington, D.C. : National Academy of Sciences,

CODEN: PNASA6; ISSN: 0027-8424

NOTE: Includes references

PUB. COUNTRY: District of Columbia; United States

DOCUMENT TYPE: Article; Conference

FILE SEGMENT: U.S. Imprints not USDA, Experiment or Extension

LANGUAGE: English

AB Agrobacterium tumefaciens is a Gram-negative, soil-borne bacterium responsible for the crown gall disease of plants. The galls result from genetic transformation of plant cells by the bacteria. Genes located on the transferred DNA (T-DNA), which is part of the large tumor-inducing (Ti) plasmid of Agrobacterium, are integrated into host plant chromosomes and expressed. This transfer requires virulence (vir) genes that map outside the T-DNA on the Ti plasmid and that encode a series of elaborate functions that appear similar to those of interbacterial plasmid transfer. It remains a major challenge to understand how T-DNA moves from Agrobacterium into the plant cell nucleus, in view of the complexity of obstacles presented by the **eukaryotic** host cell. Specific **anchoring** of bacteria to the outer surface of the plant cell seems to be an important prelude to the mobilization of the T-DNA/protein complex from the bacterial cell to the plant cell. However, the precise mode of infection is not clear, although a requirement of wounded cells has been documented. By using a microinjection approach, we show here that the process of T-DNA transfer from the bacteria to the eukaryotic nucleus

can occur entirely inside the plant cell. Such transfer is absolutely dependent on induction of vir genes and a functional virB operon. Thus, *A. tumefaciens* can function as an intracellular infectious agent in plants.

L19 ANSWER 11 OF 12 BIOTECHNO COPYRIGHT 2005 Elsevier Science B.V. on STN  
DUPLICATE

ACCESSION NUMBER: 1992:22188149 BIOTECHNO  
TITLE: Isoprenoid modification of proteins distinct from  
membrane acyl proteins in the prokaryote *Acholeplasma*  
*laidlawii*  
AUTHOR: Nystrom S.; Wieslander A.  
CORPORATE SOURCE: Department of Biochemistry, University of Umea, S-901  
87 Umea, Sweden.  
SOURCE: *Biochimica et Biophysica Acta - Biomembranes*, (1992),  
1107/1 (39-43)  
CODEN: BBBMBS ISSN: 0005-2736  
DOCUMENT TYPE: Journal; Article  
COUNTRY: Netherlands  
LANGUAGE: English  
SUMMARY LANGUAGE: English

AN 1992:22188149 BIOTECHNO

AB Isoprenylation is an important posttranslational modification that affects the activity, subunit interactions and membrane **anchoring** of different **eukaryotic** proteins. The small, cell-wall-less prokaryote *Acholeplasma laidlawii* has more than 20 membrane acyl-proteins enriched in myristoyl and palmitoyl chains. Radioactive mevalonate, a precursor to isoprenoids, was incorporated into several specific membrane proteins of 20 to 45 kDa and two soluble proteins of 23-25 kDa, respectively. No acyl proteins and none of the polar acyl lipids became labelled but these are all labelled by radioactive fatty acids. Mevalonate was incorporated mainly into a minor neutral, non-saponifiable lipid which migrated just above a C.sub.3.sub.0-isoprenoid (squalene) on TLC-plates. The isoprenoid chains could not be released by mild alkaline hydrolysis from most of the isoprenylated proteins, although this procedure releases acyl chains from lipids and all acylated proteins. Isoprenylated proteins were enriched in the detergent phase upon partition with the non-ionic detergent Triton X-114. This behaviour is similar to the acyl proteins of this organism and indicates that the isoprenoid chains give the proteins a hydrophobic character.

L19 ANSWER 12 OF 12 BIOTECHNO COPYRIGHT 2005 Elsevier Science B.V. on STN

ACCESSION NUMBER: 1988:18199299 BIOTECHNO  
TITLE: Glycolipid reanchoring of T-lymphocyte surface antigen  
CD8 using the 3' end sequence of decay-accelerating  
factor's mRNA  
AUTHOR: Tykocinski M.L.; Shu H.-K.; Ayers D.J.; Walter E.I.;  
Getty R.R.; Groger R.K.; Hauer C.A.; Medof M.E.  
CORPORATE SOURCE: Institute of Pathology, Case Western Reserve  
University, Cleveland, OH 44106, United States.  
SOURCE: *Proceedings of the National Academy of Sciences of the*  
*United States of America*, (1988), 85/10 (3555-3559)  
CODEN: PNASA6 ISSN: 0027-8424  
DOCUMENT TYPE: Journal; Article  
COUNTRY: United States  
LANGUAGE: English  
SUMMARY LANGUAGE: English

AN 1988:18199299 BIOTECHNO

AB Decay-accelerating factor (DAF) is one of a family of cell-associated proteins that undergo posttranslational modifications in which glycolipid anchoring structures are substituted for membrane-spanning sequences. The signals that direct the covalent substitution reaction in these proteins are unknown. Human DAF was expressed in Chinese hamster ovary (CHO) cells and murine BW lymphocytes. In both cases, the xenogeneic DAF in

transfectants incorporated a glycolipid anchor. A chimeric CD8-DAF cDNA, encompassing the extra-cellular region of the T-lymphocyte surface antigen CD8 and the 3' end of DAF mRNA (encoding the C-terminal region of mature DAF as well as the hydrophobic extension peptide), was expressed in human leukemia lines after transfection with an Epstein-Barr virus-based episomal vector. The chimeric protein in transfectants demonstrated glycolipid anchoring, whereas unaltered CD8 in control experiments did not. The signals directing glycolipid **anchoring** in **eukaryotic** cells are thus evolutionarily conserved and contained in the 3' end of the DAF sequence.

L9 ANSWER 1 OF 2 BIOTECHNO COPYRIGHT 2005 Elsevier Science B.V. on STN

ACCESSION NUMBER: 2002:34413748 BIOTECHNO

TITLE: In vivo selectively infective phage as a tool to detect protein interactions: Evaluation of a novel vector system with yeast Ste7p-Fus3p interacting proteins

AUTHOR: Hertveldt K.; Robben J.; Volckaert G.

CORPORATE SOURCE: K. Hertveldt, Laboratorium voor Gentechologie, Kasteelpark Arenberg 21, B-3001 Leuven, Belgium. E-mail: Kirsten.Hertveldt@agr.kuleuven.ac.be

SOURCE: Yeast, (2002), 19/6 (499-508), 31 reference(s)

CODEN: YESTE3 ISSN: 0749-503X

DOCUMENT TYPE: Journal; Article

COUNTRY: United Kingdom

LANGUAGE: English

SUMMARY LANGUAGE: English

AN 2002:34413748 BIOTECHNO

AB The selectively infective phage (SIP) approach allows rapid identification of interacting proteins by linking protein-protein interaction to phage infectivity. Infection of *E. coli* by filamentous phage depends on viral g3p. This protein consists of three domains, N1, N2 and CT. Phages lacking the N1 domain are non-infective unless a bait (X)-prey (Y) interaction links it to phage anchored N2-CT domains. We have developed all the vectors required for an in vivo selectively infective phage strategy (SIP). This includes a bait vector, pG3N1, a prey vector, pHOS41, and a gene III deletion helper phage, HPd3. The bait vector pG3N1 allows expression of a bait protein (X) fused to the C-terminus of the N1 domain. The prey vector pHOS41 allows expression of type (Y) proteins, fused to the N-terminus of the N2-CT domains. The gene III deletion helper phage delivers all phage proteins necessary for phage production, except g3p. *Escherichia coli* transformed with these three vectors produces non-infective phages unless a bait-prey interaction links the g3p domains. Fus3p and Ste7p, two proteins from the *Saccharomyces cerevisiae* pheromone-responsive pathway have been cloned to evaluate the SIP strategy. The presence of the interacting N1-Fus3p adapter increased the infectivity of Ste7p-N2-CT phages approx. 1400-fold, which makes SIP a promising technology for the detection and further investigation of interacting proteins. Copyright .COPYRG. 2002 John Wiley & Sons, Ltd.

L9 ANSWER 2 OF 2 LIFESCI COPYRIGHT 2005 CSA on STN

ACCESSION NUMBER: 2002:69230 LIFESCI

TITLE: In vivo selectively infective phage as a tool to detect protein interactions: Evaluation of a novel vector system with yeast Ste7p-Fus3p interacting proteins

AUTHOR: Hertveldt, K.; Robben, J.; Volckaert, G.

CORPORATE SOURCE: Laboratorium voor Gentechologie, Kasteelpark Arenberg 21, B-3001 Leuven, Belgium; E-mail: Kirsten.Hertveldt@agr.kuleuven.ac.be

SOURCE: Yeast, (2002) 40(6) vol. 19, no. 6, pp. 499-508.

ISSN: 0749-503X.

DOCUMENT TYPE: Journal

FILE SEGMENT: K

LANGUAGE: English

SUMMARY LANGUAGE: English

AB The selectively infective phage (SIP) approach allows rapid identification of interacting proteins by linking protein-protein interaction to phage infectivity. Infection of *E. coli* by filamentous phage depends on viral g3p. This protein consists of three domains, N1, N2 and CT. Phages lacking the N1 domain are non-infective unless a

bait (X)-prey (Y) interaction links it to phage anchored **N2-CT domains**. We have developed all the vectors required for an in vivo selectively infective phage strategy (SIP). This includes a bait vector, pG3N1, a prey vector, pHOS41, and a **gene III** deletion helper phage, HPd3. The bait vector pG3N1 allows expression of a bait protein (X) fused to the C-terminus of the N1 **domain**. The prey vector pHOS41 allows expression of prey (Y) proteins, fused to the N-terminus of the **N2-CT domains**. The **gene III** deletion helper phage delivers all phage proteins necessary for phage production, except g3p. Escherichia coli transformed with these three vectors produces non-infective phages unless a bait-prey interaction links the g3p **domains**. Fus3p and Ste7p, two proteins from the Saccharomyces cerevisiae pheromone-responsive pathway have been cloned to evaluate the SIP strategy. The presence of the interacting N1-Fus3p adapter increased the infectivity of Ste7p-**N2-CT** phages approximately 1400-fold, which makes SIP a promising technology for the detection and further investigation of interacting proteins.

=> N2 and phage and (Vh or Vl or heavy chain or light chain)

```
L10      0 FILE AGRICOLA
L11      0 FILE BIOTECHNO
L12      0 FILE CONFSCI
L13      0 FILE HEALSAFE
L14      0 FILE IMSDRUGCONF
L15      0 FILE LIFESCI
L16      0 FILE MEDICONF
L17      0 FILE PASCAL
```

TOTAL FOR ALL FILES

```
L18      0 N2 AND PHAGE AND (VH OR VL OR HEAVY CHAIN OR LIGHT CHAIN)
```

=> N2 and (Vh or Vl or heavy chain or light chain)

```
L19      1 FILE AGRICOLA
L20      9 FILE BIOTECHNO
L21      0 FILE CONFSCI
L22      0 FILE HEALSAFE
L23      0 FILE IMSDRUGCONF
L24      5 FILE LIFESCI
L25      0 FILE MEDICONF
L26      8 FILE PASCAL
```

TOTAL FOR ALL FILES

```
L27      23 N2 AND (VH OR VL OR HEAVY CHAIN OR LIGHT CHAIN)
```

=> l27 and domain

```
L28      0 FILE AGRICOLA
L29      0 FILE BIOTECHNO
L30      0 FILE CONFSCI
L31      0 FILE HEALSAFE
L32      0 FILE IMSDRUGCONF
L33      0 FILE LIFESCI
L34      0 FILE MEDICONF
L35      0 FILE PASCAL
```

TOTAL FOR ALL FILES

```
L36      0 L27 AND DOMAIN
```

=> dup rem

ENTER L# LIST OR (END):L27

DUPLICATE IS NOT AVAILABLE IN 'IMSDRUGCONF, MEDICONF'.

ANSWERS FROM THESE FILES WILL BE CONSIDERED UNIQUE

PROCESSING COMPLETED FOR L27

L37 19 DUP REM L27 (4 DUPLICATES REMOVED)

=> l27 and multi

L38 0 FILE AGRICOLA  
L39 0 FILE BIOTECHNO  
L40 0 FILE CONFSCI  
L41 0 FILE HEALSAFE  
L42 0 FILE IMSDRUGCONF  
L43 0 FILE LIFESCI  
L44 0 FILE MEDICONF  
L45 0 FILE PASCAL

TOTAL FOR ALL FILES

L46 0 L27 AND MULTI

=> d l27 ibib abs total

L27 ANSWER 1 OF 23 AGRICOLA Compiled and distributed by the National Agricultural Library of the Department of Agriculture of the United States of America. It contains copyrighted materials. All rights reserved.  
(2005) on STN

ACCESSION NUMBER: 2004:22599 AGRICOLA

DOCUMENT NUMBER: IND43627006

TITLE: ATPase activity, surface hydrophobicity, sulfhydryl content and protein degradation in refrigerated seabass muscle in modified atmosphere packaging.

AUTHOR(S): Masniyom, P.; Benjakul, S.; Visessanguan, W.

AVAILABILITY: DNAL (TX545.J6)

SOURCE: Journal of food biochemistry, 2004 Feb. Vol. 28, no. 1  
p. 43-60  
ISSN: 0145-8884

NOTE: Includes references

DOCUMENT TYPE: Article

FILE SEGMENT: Other US

LANGUAGE: English

AB The effect of modified atmosphere packaging (80% CO<sub>2</sub>, 10% O<sub>2</sub>, 10% N<sub>2</sub>) on ATPase activity, surface hydrophobicity, sulfhydryl content and degradation of proteins in seabass muscle during storage at 4C was investigated. No changes in Ca<sup>2+</sup>-, Mg<sup>2+</sup>-, Mg<sup>2+</sup>-Ca<sup>2+</sup>-ATPase activities of natural actomyosin (NAM) in seabass slices kept under MAP were observed throughout the storage for up to 21 days (P > 0.05). However, a slightly increased Mg<sup>2+</sup>-EGTA-ATPase was found. For seabass slices stored under air atmosphere, Ca<sup>2+</sup>-ATPase activity decreased, whereas Mg<sup>2+</sup>-EGTA-ATPase activity increased (P < 0.05) with a concomitant loss in Ca<sup>2+</sup>-sensitivity. Lower decreases in total sulfhydryl content but higher increases in surface hydrophobicity were observed in samples stored under MAP, compared to those kept under air atmosphere. No marked autolytic degradation in samples kept under MAP was observed throughout the storage as monitored by no changes in myosin **heavy chain**, free  $\alpha$ -amino acid and trichloroacetic acid soluble peptide. Conversely, a considerable degradation was found in samples kept under air atmosphere, especially after 9 days of storage. Therefore, MAP is a promising means to retard the changes in muscle proteins, especially degradation.

L27 ANSWER 2 OF 23 BIOTECHNO COPYRIGHT 2005 Elsevier Science B.V. on STN

ACCESSION NUMBER: 2002:35304325 BIOTECHNO

TITLE: Antibody epitopes on the neuraminidase of a recent H3N2 influenza virus (A/Memphis/31/98)

AUTHOR: Gulati U.; Hwang C.-C.; Venkatramani L.; Gulati S.; Stray S.J.; Lee J.T.; Laver W.G.; Bochkarev A.; Zlotnick A.; Air G.M.

CORPORATE SOURCE: G.M. Air, Department of Biochemistry, Univ. of Oklahoma Hlth. Sci. Center, Oklahoma City, OK 73104,



United States.  
E-mail: gillian-air@ouhsc.edu  
SOURCE: Journal of Virology, (2002), 76/23 (12274-12280), 45  
reference(s)  
CODEN: JOVIAM ISSN: 0022-538X  
DOCUMENT TYPE: Journal; Article  
COUNTRY: United States  
LANGUAGE: English  
SUMMARY LANGUAGE: English  
AN 2002:35304325 BIOTECHNO  
AB We have characterized monoclonal antibodies raised against the  
neuraminidase (NA) of a Sydney-like influenza virus (A/Memphis/31/98,  
H3N2) in a reassortant virus A/NWS/33.sub.H.sub.A-A/Mem/31/98.sub.N.sub.A  
(H1N2) and nine escape mutants selected by these monoclonal antibodies.  
Five of the antibodies use the same **heavy chain** VDJ  
genes and may not be independent. Another antibody, Mem5, uses the same  
V.sub.H and J genes with a different D gene and different isotype.  
Sequence changes in escape mutants selected by these antibodies occur in  
two loops of the NA, at amino acid 198, 199, 220, or 221. These amino  
acids are located on the opposite side of the NA monomer to the major  
epitopes found in N9 and early **N2** NAs. Escape mutants with a  
change at 198 have reduced NA activity compared to the wild-type virus.  
Asp198 points toward the substrate binding pocket, and we had previously  
found that a site-directed mutation of this amino acid resulted in a loss  
of enzyme activity (M. R. Lentz, R. G. Webster, and G. M. Air,  
Biochemistry 26:5351-5358, 1987). Mutations at residue 199, 220, or 221  
did not alter the NA activity significantly compared to that of wild-type  
NA. A 3.5-Å structure of Mem5 Fab complexed with the Mem/98 NA shows  
that the Mem5 antibody binds at the sites of escape mutation selected by  
the other antibodies.

L27 ANSWER 3 OF 23 BIOTECHNO COPYRIGHT 2005 Elsevier Science B.V. on STN  
ACCESSION NUMBER: 1999:29537832 BIOTECHNO  
TITLE: Mapping of a gene responsible for dermatitis in NOA  
(Naruto Research Institute Otsuka Atrichia) mice, an  
animal model of allergic dermatitis  
AUTHOR: Natori K.; Tamari M.; Watanabe O.; Onouchi Y.;  
Shiomoto Y.; Kubo S.; Nakamura Y.  
CORPORATE SOURCE: Y. Nakamura, Laboratory of Molecular Medicine,  
Institute of Medical Science, University of Tokyo,  
4-6-1 Shirokanedai, Minato-ku, Tokyo 108-8639, Japan.  
E-mail: yusuke@ims.u-tokyo.ac.jp  
SOURCE: Journal of Human Genetics, (1999), 44/6 (372-376), 27  
reference(s)  
CODEN: JHGEFR ISSN: 1434-5161  
DOCUMENT TYPE: Journal; Article  
COUNTRY: Japan  
LANGUAGE: English  
SUMMARY LANGUAGE: English  
AN 1999:29537832 BIOTECHNO  
AB The NOA (Naruto Research Institute Otsuka Atrichia) mouse is an animal  
model of allergic or atopic dermatitis, a condition characterized by  
ulcerative skin lesions with accumulation of mast cells and increased  
serum IgE. These features of the murine disease closely resemble human  
atopy and atopic disorders. We performed linkage analysis in NOA  
back-cross progeny, as a step toward identifying and isolating a gene  
responsible for the NOA phenotype. We crossed NOA mice with five other  
murine strains (C57BL/6J, IQI, C3H/HeJ, DBA/2J, and BALB/cByJ) and then  
bred back-cross animals. Using microsatellite markers, we scanned the  
entire genomes of 559 **N2** offspring from the five parental  
strains. Linkage analysis revealed a significant association between  
ulcerative skin lesions and markers on murine chromosome 14. Statistical  
analysis indicated that the critical region was assigned to the vicinity

of D14Mit236 and D14Mit160.

L27 ANSWER 4 OF 23 BIOTECHNO COPYRIGHT 2005 Elsevier Science B.V. on STN  
ACCESSION NUMBER: 1996:27068406 BIOTECHNO

TITLE: Purification and sequencing of yellow mustard seed  
napin small and large chains that are phosphorylated  
by plant calcium-dependent protein kinase and are  
calmodulin antagonists

AUTHOR: Neumann G.M.; Condrón R.; Polya G.M.

CORPORATE SOURCE: G.M. Polya, School of Biochemistry, La Trobe  
University, Bundoora, Vic. 3083, Australia.

SOURCE: Plant Science, (1996), 119/1-2 (49-66), 44  
reference(s)

CODEN: PLSCE4 ISSN: 0168-9452

PUBLISHER ITEM IDENT.: S0168945296044767

DOCUMENT TYPE: Journal; Article

COUNTRY: Ireland

LANGUAGE: English

SUMMARY LANGUAGE: English

AN 1996:27068406 BIOTECHNO

AB A multiplicity of small (S) and large (L) napin subunits were purified from yellow mustard (*Sinapis alba* L.) seeds by a protocol involving extraction, successive batch-wise cation exchange chromatography on carboxymethylcellulose (CM52), cation exchange HPLC on an SP5PW column and reversed phase HPLC on a C18 column. Initial cation exchange HPLC resolved 4 major zones of proteins (M1, M2, N1 and N2) that can be phosphorylated by plant Ca<sup>sup.2.sup.</sup>-dependent protein kinase (CDPK). Electrospray ionization mass spectrometry (ESMS) revealed that M1 and M2 are 6 kDa proteins, later identified as  $\gamma$ -thionin-related proteins. ESMS of fractions N1 and N2 revealed the presence of 14.5 kDa proteins identified as napin complexes, each composed of a single small subunit linked to a single large subunit and involving 4 disulphide linkages. The napin complexes (N1A, N1B, N1C, N1D, N2A, N2B and N2C) were disrupted and the constituent small subunits (S1, S2 and S3) and large subunits (L1A, L1B, L1C, L2A, L2B and L2C) were resolved by reversed phase HPLC and precise average molecular masses determined by ESMS. The small and large subunits have average molecular masses of about 4.4 kDa and 10.1 kDa, respectively. The masses of each napin complex can be precisely accounted for from the masses of the constituent subunits. Thus the major complex N2A (14 569  $\pm$  3 Da) is evidently composed of S3 (4434.0  $\pm$  1.5 Da) and L2A (10 142.5  $\pm$  1.5 Da) and involves 4 disulphides (loss of 8.0 Da), the expected mass of S3 + L2A-8H being 14 569  $\pm$  2 Da. The yellow mustard napin large chain L2A is phosphorylated by wheat CDPK on Ser<sup>sup.6.sup.</sup> within the sequence LQHVIS<sup>sup.6.sup.</sup>ORIIY. The complete sequence of this and other large (and small) napin subunits were determined from Edman sequencing and/or ESMS data by comparison with published napin sequences. Yellow mustard seed CM52- binding fractions decrease the Ca<sup>sup.2.sup.</sup>-dependent fluorescence emission of dansyl-CaM and yellow mustard small and large chains inhibit CaM-dependent myosin **light chain** kinase (MLCK).

L27 ANSWER 5 OF 23 BIOTECHNO COPYRIGHT 2005 Elsevier Science B.V. on STN  
ACCESSION NUMBER: 1996:27041593 BIOTECHNO

TITLE: B-1a, B-1b and conventional B cell lymphoma from  
enzootic bovine leukosis

AUTHOR: Wu D.; Takahashi K.; Murakami K.; Tani K.; Koguchi A.;  
Asahina M.; Goryo M.; Aida Y.; Okada K.

CORPORATE SOURCE: K. Okada, Dept. of Veterinary Pathology, Faculty of  
Agriculture, Iwate University, Morioka 020, Japan.

SOURCE: Veterinary Immunology and Immunopathology, (1996),  
55/1-3 (63-72), 20 reference(s)

CODEN: VIIMDS ISSN: 0165-2427

PUBLISHER ITEM IDENT.: S0165242796056310  
DOCUMENT TYPE: Journal; Article  
COUNTRY: Netherlands  
LANGUAGE: English  
SUMMARY LANGUAGE: English

AN 1996:27041593 BIOTECHNO

AB In order to characterize the phenotypes of tumor cells and to clarify from which B cell lineage the lymphomas were derived, ten cows with enzootic bovine leukosis were examined by means of immunohistologic staining and flow cytometry. The tumor cells expressed mainly major histocompatibility complex (MHC) class II.sup.+ (10/10), BoCD11b.sup.+ (9/10), IgG.sub.1.sup.+ (8/10), B-B2.sup.+ (8/10) BoCD5.sup.+ (7/10), and  $\lambda$  light chain.sup.+ (7/10). Tumor cells from only one animal expressed sIgM.sup.+ (1/10). Tumor cells from all ten animals were negative for IgG.sub.2, BoCD3, BoCD4, BoCD8, WC1-N2, and IL-2R $\alpha$ . The phenotypes of these tumor cells were all slightly different, suggesting that bovine leukemia virus (BLV)-induced lymphoma expresses phenotypic diversity. Moreover, tumor cells from seven cattle coexpressed BoCD5 and BoCD11b (B-1a cells). On the other hand, tumor cells from two of them only expressed BoCD11b (B-1b cells), and those from one were negative for both BoCD5 and BoCD11b (conventional B cells). Therefore, we concluded that BLV-induced lymphoma cells can be derived from B-1a, B-1b and conventional B cells.

L27 ANSWER 6 OF 23 BIOTECHNO COPYRIGHT 2005 Elsevier Science B.V. on STN

ACCESSION NUMBER: 1996:26308902 BIOTECHNO

TITLE: Molecular analysis of the leukaemic B cell in adult and childhood acute lymphoblastic leukaemia

AUTHOR: Coyle L.A.; Papaioannou M.; Yaxley J.C.; Chim J.S.; Attard M.; Hoffbrand A.V.; Foroni L.

CORPORATE SOURCE: Department of Haematology, Royal Free Hospital, School of Medicine, Pond Street, London NW3 2QG, United Kingdom.

SOURCE: British Journal of Haematology, (1996), 94/4 (685-693)

CODEN: BJHEAL ISSN: 0007-1048

DOCUMENT TYPE: Journal; Article

COUNTRY: United Kingdom

LANGUAGE: English

SUMMARY LANGUAGE: English

AN 1996:26308902 BIOTECHNO

AB Immunoglobulin **heavy chain** gene (IgH gene) rearrangements are found in the majority of cases of B-lineage acute lymphoblastic leukaemia (ALL). We have examined bone marrow samples taken at presentation or relapse from 109 patients (79 adults and 30 children) and have performed sequence analysis of the complementarity determining region 3 (CDR3) on 65 alleles from 54 patients. We aimed to define immunoglobulin **heavy chain** (IgH) variable segment family use and investigate biological and structural features of the B cell in adult and childhood ALL. Using the FR1 fingerprinting method, a rearranged band was identified in 70 (89%) of 79 adult ALL and in 29 (9.7%) of 30 childhood ALL. This study found no preferential use or selection of IgH **VH** genes and no statistically significant structural differences between normal and leukaemic B cells in either adult and childhood ALL. An equal proportion of amplifiable cases of adult and childhood ALL uses more than one **VH** family gene (24/70, 34%, and 8/29, 27.5%, respectively). There were no significant differences in the structure or size of the CDR3 region and the variable (V) or joining (J) segment use in ALL patients compared to normal B cells. We observed that the **N2** region was shorter than N1 in children whereas the opposite was observed in adults (not statistically significant). The J4 segment was a more common rearrangement in children than in adults, and in both groups J4 was more frequently associated with multiple D segment VDJ rearrangements. An increase in VH6 use in

leukaemic alleles compared to normal B lymphocytes (2%) was observed but it was not statistically significant in our group of patients. Amongst children and adults, in-frame CDR3 junctions occurred in 78% and 64% of rearranged alleles, respectively, compared to 75% of in-frame sequences reported by others to occur among normal 13 cells.

L27 ANSWER 7 OF 23 BIOTECHNO COPYRIGHT 2005 Elsevier Science B.V. on STN  
ACCESSION NUMBER: 1983:13135608 BIOTECHNO  
TITLE: Some T-cell leukemia lines express surface markers related to a restricted set of human V(H) determinants  
AUTHOR: Marchalonis J.J.; Vasta G.R.; Hunt J.C.; et al.  
CORPORATE SOURCE: Dep. Biochem., Med. Univ. South Carolina, Charleston, SC 29425, United States.  
SOURCE: Cellular Immunology, (1983), 77/1 (161-175)  
CODEN: CLIMB8  
DOCUMENT TYPE: Journal; Article  
COUNTRY: United States  
LANGUAGE: English

AN 1983:13135608 BIOTECHNO

AB Serological studies were carried out to obtain information regarding the relationship of the V(H)-related determinants expressed by certain permanent in vitro T-cell leukemia lines and corresponding determinants expressed by characterized human serum immunoglobulins. A panel of conventional (goat and rabbit) antisera, produced against various Fab-related fragments of monoclonal human Waldenstrom macroglobulins and polyclonal IgG molecules, bound to certain in vitro T-cell leukemia lines, notably, 70-N2, MT-1, YT4E, and HUT78, as shown by microhemagglutinin. Inhibition studies using characterized myeloma proteins to inhibit this agglutination indicated the expression of a restricted V(H)-related determinant by these T-cell lines. Parallel studies performed using conventional (rabbit) and murine monoclonal/hybridoma antibodies produced against the isolated 68,000-Da V(H)-related product synthesized by the 70-N2 line showed that the determinant expressed by this molecule was restricted in expression, comprising 2-3% of the normal, polyclonal human Fab pool, and that the determinants found on the other positive T-cell leukemias were cross-reactive rather than identical. The inhibition studies suggest that the determinant resides between residue 22 and the end of the V(H) region. These results further define the antigenic nature of the V(H)-related marker found on the surfaces of certain normal and neoplastic T-cell lines.

L27 ANSWER 8 OF 23 BIOTECHNO COPYRIGHT 2005 Elsevier Science B.V. on STN  
ACCESSION NUMBER: 1982:12028840 BIOTECHNO  
TITLE: Two types of calcium-dependent protein phosphorylations modulated by calmodulin antagonists. Naphthalenesulfonamide derivatives  
AUTHOR: Tanaka T.; Ohmura T.; Yamakado T.; Hidaka H.  
CORPORATE SOURCE: Dep. Pharmacol., Mie Univ. Sch. Med., Edobashi, Tsu 514, United States.  
SOURCE: Molecular Pharmacology, (1982), 22/2 (408-412)  
CODEN: MOPMA3  
DOCUMENT TYPE: Journal; Article  
COUNTRY: United States  
LANGUAGE: English

AN 1982:12028840 BIOTECHNO

AB Ca.sup.2+.sup.+-dependent protein phosphorylations activated by calmodulin or phospholipid were studied using selective inhibitors. Both protein phosphorylations were inhibited by N-(6-aminoethyl)-5-chloro-1-naphthalenesulfonamide (W-7) and its derivatives. Kinetic analysis indicated that the primary effect of these agents was mediated through a competitive inhibition of enzyme activation by interaction with calmodulin or phospholipid, and K(i) values of W-7 for

calmodulin-dependent phosphorylation and phospholipid-dependent protein kinase were 12  $\mu$ M and 110  $\mu$ M, respectively. The addition of Ca<sup>2+</sup> inhibited the binding of  $\epsilon$ -[3H]W-7 to phosphatidylserine but not the binding to calmodulin. The potencies of naphthalenesulfonamide derivatives as inhibitors of Ca<sup>2+</sup>, calmodulin-dependent protein kinase were dependent on the length of the alkyl chain (C<sub>2</sub>-C<sub>10</sub>) but not on Ca<sup>2+</sup>-activated, phospholipid-dependent protein kinase. These results suggest that naphthalenesulfonamide derivatives may be more selective inhibitors of Ca<sup>2+</sup>, calmodulin-dependent protein phosphorylation than is Ca<sup>2+</sup>-activated, phospholipid-dependent protein kinase and that the mechanism of interaction between W-7 and phosphatidylserine differs from the interaction between W-7 and calmodulin. These agents are useful tools for elucidating the physiological role of Ca<sup>2+</sup>-dependent protein phosphorylation.

L27 ANSWER 9 OF 23 BIOTECHNO COPYRIGHT 2005 Elsevier Science B.V. on STN  
 ACCESSION NUMBER: 1979:10193547 BIOTECHNO  
 TITLE: Effect of novel specific myosin **light chain** kinase inhibitors on Ca<sup>2+</sup>-activated Mg<sup>2+</sup>-ATPase of chicken gizzard actomyosin  
 AUTHOR: Hidaka H.; Naka M.; Yamaki T.  
 CORPORATE SOURCE: Dept. Pharmacol., Mie Univ. Sch. Med., Tsu, Japan.  
 SOURCE: Biochemical and Biophysical Research Communications, (1979), 90/3 (694-699)  
 CODEN: BBRCA0  
 DOCUMENT TYPE: Journal; Article  
 COUNTRY: United States  
 LANGUAGE: English  
 AN 1979:10193547 BIOTECHNO

L27 ANSWER 10 OF 23 BIOTECHNO COPYRIGHT 2005 Elsevier Science B.V. on STN  
 ACCESSION NUMBER: 1980:10176274 BIOTECHNO  
 TITLE: Calcium-regulated modulator protein interacting agents inhibit smooth muscle calcium-stimulated protein kinase and ATPase  
 AUTHOR: Hidaka H.; Yamaki T.; Naka M.; et al.  
 CORPORATE SOURCE: Dept. Pharmacol., Sch. Med., Mie Univ., Tsu, Japan.  
 SOURCE: Molecular Pharmacology, (1980), 17/1 (66-72)  
 CODEN: MOPMA3  
 DOCUMENT TYPE: Journal; Article  
 COUNTRY: United States  
 LANGUAGE: English  
 AN 1980:10176274 BIOTECHNO  
 AB Reagents such as N-(6-aminohexyl)-5-chloro-1-naphthalenesulfonamide (W-7), chlorpromazine, prenylamine, and N<sup>2</sup>-dansyl-L-arginine-4-t-butylpiperidine amide (Number 233) that interact with Ca<sup>2+</sup>-regulated modulator protein (modulator protein, calmodulin) were found to inhibit dose dependently not only Ca<sup>2+</sup>-dependent ATPase of chicken gizzard actomyosin. Inhibition of Ca<sup>2+</sup>-dependent ATPase by these agents was prevented by the addition of modulator protein. These agents did not inhibit calcium-independent Mg<sup>2+</sup>-ATPase of actomyosin. Ca<sup>2+</sup>-dependent transfer of  $\gamma$ -[3<sup>2</sup>P]ATP to the 20,000-dalton **light chain** of the gizzard myosin in the presence of Mg<sup>2+</sup> was also inhibited dose dependently by these agents. The concentrations of these agents producing 50% inhibition of the Ca<sup>2+</sup>-dependent ATPase activity were found to be similar to concentrations producing 50% inhibition of myosin **light chain** phosphorylation, thereby suggesting that the inhibition of Ca<sup>2+</sup>-dependent ATPase of actomyosin by these drugs is due to their inhibition of myosin **light chain** phosphorylation. W-7 bound to

Ca.sup.2.sup.+ modulator protein complex, but not to the modulator protein in the presence of EGTA. Number 233 and chlorpromazine inhibited the binding of W-7 to the Ca.sup.2.sup.+modulator complex, suggesting that Number 233 and chlorpromazine bind to modulator protein. The modulator protein has two classes of W-7 binding sites: three functional sites with a high affinity for W-7 ( $K(w-7) = 11 \mu M$ ) and nine sites with a low affinity for the drug ( $K(w-7) = 200 \mu M$ ). W-7 did not show a significant binding to actin, myosin, tropomyosin, and bovine serum albumin at the concentration of the drug capable of binding to modulator protein. Troponin C was the only protein other than modulator protein that bound W-7 significantly but the affinity ( $K(w-7) = 25 \mu M$ ) of this protein for W-7 was lower than that of modulator protein. These results suggest that agents that interact with modulator protein produce relaxation of smooth muscle by inhibition of modulator protein-dependent myosin **light chain** phosphorylation thus suppressing the actin-myosin interaction and concomitant myosin ATPase activation.

L27 ANSWER 11 OF 23 LIFESCI COPYRIGHT 2005 CSA on STN

ACCESSION NUMBER: 2005:21910 LIFESCI

TITLE: Transcriptional Regulation and Life-span Modulation of Cytosolic Aconitase and Ferritin Genes in *C.elegans*  
AUTHOR: Kim, Young-Il; Cho, Jeong Hoon; Yoo, Ook Joon; Ahnn, Joohong

CORPORATE SOURCE: Department of Life Science, Gwangju Institute of Science and Technology, 1 Oryoung-dong, Buk-gu, Gwangju, 500-712, South Korea; E-mail: joohong@gist.ac.kr

SOURCE: Journal of Molecular Biology [J. Mol. Biol.], (20040910) vol. 342, no. 2, pp. 421-433.  
ISSN: 0022-2836.

DOCUMENT TYPE: Journal

FILE SEGMENT: N

LANGUAGE: English

SUMMARY LANGUAGE: English

AB Ferritin is the major iron storage protein regulating cytosolic concentration of iron by storing excess iron. Vertebrate ferritins are heteropolymeric proteins composed of **heavy chain** and **light chain** subunits. We have characterized two *Caenorhabditis elegans* genes (*ftn-1* and *ftn-2*), which encode ferritin homologs showing high degree of similarity to mammalian ferritin **heavy chains**. Even though these two ferritins are more than 78% identical in amino acid sequence, our data show that expression patterns and responses to iron are quite different. Cytosolic aconitase (*aco-1*), iron regulatory protein, is known to regulate cellular iron concentration by modulating translation of the ferritin mRNA in addition to its enzymatic activity that converts citrate into iso-citrate. We have shown that the expression levels of *aco-1* and *ftn-1* genes are both regulated by iron treatment but in opposite ways. Interestingly, mutant animals lacking *ACO-1* and *FTN-1* show significantly reduced life-span upon iron stress, while **N2** and *ftn-2* animals show no difference. Our results suggest that *ftn-1* and *aco-1* are transcriptionally regulated by iron and are important for iron homeostasis affecting life-span upon iron stress conditions in *C.elegans*.

L27 ANSWER 12 OF 23 LIFESCI COPYRIGHT 2005 CSA on STN

ACCESSION NUMBER: 2003:5922 LIFESCI

TITLE: Antibody Epitopes on the Neuraminidase of a Recent H3N2 Influenza Virus (A/Memphis/31/98)

AUTHOR: Gulati, U.; Hwang, C.; Venkatramani, L.; Gulati, S.; Stray, S.J.; Lee, J.T.; Laver, W.G.; Bochkarev, A.; Zlotnick, A.; Air, G.M.\*

CORPORATE SOURCE: Department of Biochemistry and Molecular Biology, University of Oklahoma Health Sciences Center, Oklahoma City, OK 73104; E-mail: gillian-air@ouhsc.edu

SOURCE: Journal of Virology [J. Virol.], (20021200) vol. 76, no. 23, pp. 12274-12280.  
ISSN: 0022-538X.

DOCUMENT TYPE: Journal  
FILE SEGMENT: N; V  
LANGUAGE: English  
SUMMARY LANGUAGE: English

AB We have characterized monoclonal antibodies raised against the neuraminidase (NA) of a Sydney-like influenza virus (A/Memphis/31/98, H3N2) in a reassortant virus A/NWS/33 sub(HA)-A/Mem/31/98 sub(NA) (H1N2) and nine escape mutants selected by these monoclonal antibodies. Five of the antibodies use the same **heavy chain** VDJ genes and may not be independent. Another antibody, Mem5, uses the same V sub(H) and J genes with a different D gene and different isotype. Sequence changes in escape mutants selected by these antibodies occur in two loops of the NA, at amino acid 198, 199, 220, or 221. These amino acids are located on the opposite side of the NA monomer to the major epitopes found in N9 and early **N2** NAs. Escape mutants with a change at 198 have reduced NA activity compared to the wild-type virus. Asp198 points toward the substrate binding pocket, and we had previously found that a site-directed mutation of this amino acid resulted in a loss of enzyme activity (M. R. Lentz, R. G. Webster, and G. M. Air, Biochemistry 26:5351-5358, 1987). Mutations at residue 199, 220, or 221 did not alter the NA activity significantly compared to that of wild-type NA. A 3.5-Aa structure of Mem5 Fab complexed with the Mem/98 NA shows that the Mem5 antibody binds at the sites of escape mutation selected by the other antibodies.

L27 ANSWER 13 OF 23 LIFESCI COPYRIGHT 2005 CSA on STN

ACCESSION NUMBER: 2000:50616 LIFESCI

TITLE: Fibre type-specific expression of p94, a skeletal muscle-specific calpain

AUTHOR: Jones, S.W.; Parr, T.; Sensky, P.L.; Scothern, G.P.; Bardsley, R.G.\*; BATTERY, P.J.

CORPORATE SOURCE: Division of Nutritional Biochemistry, School of Biological Sciences, University of Nottingham, Sutton Bonington Campus, Loughborough, Leicestershire, LE12 5RD, UK; E-mail: ronald.bardsley@nott.ac.uk

SOURCE: Journal of Muscle Research and Cell Motility [J. Muscle Res. Cell Motil.], (19990500) vol. 20, no. 4, pp. 417-424.  
ISSN: 0142-4319.

DOCUMENT TYPE: Journal  
FILE SEGMENT: T  
LANGUAGE: English  
SUMMARY LANGUAGE: English

AB Members of the calpain proteinase family are present in all mammalian cells, although a novel calpain 94 kDa isoform is found almost exclusively in skeletal muscle. p94 is difficult to purify from muscle and recombinant p94 autolyses rapidly when expressed in COS cells. However, in vivo the enzyme may be stabilised by interaction with titin, which has two well-characterised binding sites for p94 at the **N2**- and M-lines. Both these titin subdomains are subject to muscle-specific alternative splicing, which could be related to p94 expression level or stability in muscles of different fibre type. In this study, porcine longissimus dorsi (LD), trapezius (TZ) and adductor longus (AL) were characterised as fast, intermediate and slow using commercially available specific anti-human fast- and slow-myosin **heavy chain** mAbs and also by conventional histochemistry. p94 was quantified both in whole muscle preparations and single fibres by western blotting using an anti-p94 antiserum generated by expressing a recombinant p94 sequence as a GST fusion protein antigen. SDS PAGE and immunoblotting revealed a single band of approximately 94 kDa with identical mobility in all muscle and fibre preparations. The intensity of the 94 kDa band was greater in LD (22 plus or minus 1.7 densitometric units mean plus or minus SEM, n = 3) than TZ

and AL (10 plus or minus 2.3 and 6 plus or minus 0.9 units, respectively). Expressed as a ratio relative to actin immunoreactivity, p94 is present in all types of single fibres isolated from TZ, but at a significantly lower level ( $P < 0.01$ ) in slow type I (0.08 plus or minus 0.01,  $n = 9$ ), compared to fast IIA/IIB fibres (0.22 plus or minus 0.02,  $n = 26$ ). No evidence was seen for rapid or variable rate of p94 degradation in either type of fibre. These data suggest a positive correlation between p94 expression level and fast glycolytic characteristics in porcine muscle.

L27 ANSWER 14 OF 23 LIFESCI COPYRIGHT 2005 CSA on STN

ACCESSION NUMBER: 97:82441 LIFESCI

TITLE: B-1a, B-1b and conventional B cell lymphoma from enzootic bovine leukosis

AUTHOR: Wu, Donglai; Takahashi, K.; Murakami, K.; Tani, K.; Koguchi, A.; Asahina, M.; Goryo, M.; Aida, Y.; Okada, K.\*

CORPORATE SOURCE: Dep. Veterinary Pathol., Fac. Agric., Iwate Univ., Morioka 020, Japan

SOURCE: VET. IMMUNOL. IMMUNOPATHOL., (1996) vol. 55, no. 1-3, pp. 63-72.

ISSN: 0165-2427.

DOCUMENT TYPE: Journal

FILE SEGMENT: V; F

LANGUAGE: English

SUMMARY LANGUAGE: English

AB In order to characterize the phenotypes of tumor cells and to clarify from which B cell lineage the lymphomas were derived, ten cows with enzootic bovine leukosis were examined by means of immunohistologic staining and flow cytometry. The tumor cells expressed mainly major histocompatibility complex (MHC) class II super(+) (10/10), BoCD11b super(+) (9/10), IgG sub(1) super(+) (8/10), B-B2 super(+) (8/10) BoCD5 super(+) (7/10), and lambda **light chain** super(+) (7/10). Tumor cells from only one animal expressed sIgM super(+) (1/10). Tumor cells from all ten animals were negative for IgG sub(2), BoCD3, BoCD4, BoCD8, WC1-N2, and IL-2R alpha. The phenotypes of these tumor cells were all slightly different, suggesting that bovine leukemia virus (BLV)-induced lymphoma expresses phenotypic diversity. Moreover, tumor cells from seven cattle coexpressed BoCD5 and BoCD11b (B-1a cells). On the other hand, tumor cells from two of them only expressed BoCD11b (B-1b cells), and those from one were negative for both BoCD5 and BoCD11b (conventional B cells). Therefore, we concluded that BLV-induced lymphoma cells can be derived from B-1a, B-1b and conventional B cells.

L27 ANSWER 15 OF 23 LIFESCI COPYRIGHT 2005 CSA on STN

ACCESSION NUMBER: 94:58165 LIFESCI

TITLE: Screening onion varieties for resistance to Thrips tabaci Lind. and Helicoverpa armigera (Hubner)

AUTHOR: Brar, K.S.; Sidhu, A.S.; Chadha, M.L.

CORPORATE SOURCE: Dep. Veg. Crops, Landscaping and Floric., Punjab Agric. Univ., Ludhiana-141 004, India

SOURCE: J. INSECT SCI., (1993) vol. 6, no. 1, pp. 123-124.

ISSN: 0970-3837.

DOCUMENT TYPE: Journal

FILE SEGMENT: Z

LANGUAGE: English

SUMMARY LANGUAGE: English

AB Sixtyone genotypes (bulb crop) were screened against Thrips tabaci Lind. and 12 varieties (bulb crop) and 37 lines (seed crop) were evaluated against Helicoverpa armigera (Hub.). Genotypes PBR 3, PBR 4, **VL** 1, Number 18, Number 19 and Pusa Ratnar were less preferred by T. tabaci. The lowest infestation of H. armigera in bulb crop was recorded on PBR 5, PBR 6, **N2-4-1**, N 53, Arka Niketan and PO I. In seed crop, one line Sel. 102-1 was free from this pest while Number 21, IHR 387, AFLR and Arka Niketan were promising.



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ACCESSION NUMBER: 2005-0163849 PASCAL  
COPYRIGHT NOTICE: Copyright .COPYRGT. 2005 INIST-CNRS. All rights reserved.  
TITLE (IN ENGLISH): Geniohyoid muscle properties and myosin **heavy chain** composition are altered after short-term intermittent hypoxic exposure  
AUTHOR: PAE Eung-Kwon; WU Jennifer; NGUYEN Daniel; MONTI Ryan; HARPER Ronald M.  
CORPORATE SOURCE: Section of Orthodontics, University of California at Los Angeles School of Dentistry, Los Angeles, California, United States; Department of Neurobiology, David Geffen School of Medicine at University of California at Los Angeles, Los Angeles, California, United States  
SOURCE: Journal of applied physiology : (1985), (2005), 98(3), 889-894, 50 refs.  
ISSN: 8750-7587 CODEN: JAPHEV  
DOCUMENT TYPE: Journal  
BIBLIOGRAPHIC LEVEL: Analytic  
COUNTRY: United States  
LANGUAGE: English  
AVAILABILITY: INIST-5400, 354000126362710180

AN 2005-0163849 PASCAL

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AB Patients with obstructive sleep apnea (OSA) often exhibit fatigued or inefficient upper airway dilator and constrictor muscles; an upper airway dilator, the geniohyoid (GH) muscle, is a particular example. Intermittent hypoxia (IH) is a frequent concomitant of OSA, and it may trigger muscle fiber composition changes that are characteristic of a fatigable nature. We examined effects of short-term IH on diaphragmatic and GH muscle fiber composition and fatigue properties by exposing 24 rats to alternating 10.3% O<sub>2</sub>-balance N<sub>2</sub> and room air every 480 s (240 s duty cycle) for a total duration of 5, 10, 15, 20, or 30 h. Sternohyoid fiber composition was also examined. Control animals were exposed to room air on the same schedule. Single-fiber analyses showed that GH muscle fiber types changed completely from myosin **heavy chain** (MHC) type 2A to MHC type 2B after 10 h of exposure, and the conversion was maintained for at least 30 h. Sternohyoid muscle fibers showed a delayed transition from MHC type 2A/2B to MHC type 2B. In contrast, major fiber types of the diaphragm were not significantly altered. The GH muscles showed similar tension-frequency relationships in all groups, but an increased fatigability developed, proportional to the duration of IH treatment. We conclude that short-term IH exposure alters GH muscle composition and physical properties toward more fatigable, fast-twitch types and that it may account for the fatigable upper airway fiber types found in sleep-disturbed breathing.

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ACCESSION NUMBER: 2004-0532238 PASCAL  
COPYRIGHT NOTICE: Copyright .COPYRGT. 2004 INIST-CNRS. All rights reserved.  
TITLE (IN ENGLISH): Remodeling of excitation-contraction coupling in transgenic mice expressing ATP-insensitive sarcolemmal K<sub>sub.A</sub> sub.T sub.P channels  
AUTHOR: FLAGG Thomas P.; CHARPENTIER Flavien; MANNING-FOX Jocelyn; REMEDI Maria Sara; ENKVETCHAKUL Decha; LOPATIN Anatoli; KOSTER Joseph; NICHOLS Colin  
CORPORATE SOURCE: Department of Cell Biology and Physiology, Washington University School of Medicine, St. Louis, Missouri

63110, United States; Institut National de la Sante et de la Recherche Medicale Unite 533, Physiopathologie et Pharmacologie Cellulaires et Moleculaires, Faculte de Medecine, 44035 Nantes, France; Department of Pharmacology, University of Alberta, Edmonton T6G 2H7, Alberta, Canada; Department of Physiology, University of Michigan Medical School, Ann Arbor, Michigan 48109, United States

SOURCE: American journal of physiology. Heart and circulatory physiology, (2004), 55(4), 1361-1369, 31 refs.

ISSN: 0363-6135 CODEN: AJPPDI

DOCUMENT TYPE: Journal

BIBLIOGRAPHIC LEVEL: Analytic

COUNTRY: United States

LANGUAGE: English

AVAILABILITY: INIST-670D, 354000113516410170

AN 2004-0532238 PASCAL

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AB Reducing the ATP sensitivity of the sarcolemmal ATP-sensitive K<sub>sup</sub>.+ (K<sub>sub</sub>.A<sub>sub</sub>.T<sub>sub</sub>.P) channel is predicted to lead to active channels in normal metabolic conditions and hence cause shortened ventricular action potentials and reduced myocardial inotropy. We generated transgenic (TG) mice that express an ATP-insensitive K<sub>sub</sub>.A<sub>sub</sub>.T<sub>sub</sub>.P channel mutant [Kir6.2(Δ N2-30,K185Q)] under transcriptional control of the α-myosin **heavy chain** promoter. Strikingly, myocyte contraction amplitude was increased in TG myocytes (15.68 ± 1.15% vs. 10.96 ± 1.49%), even though K<sub>sub</sub>.A<sub>sub</sub>.T<sub>sub</sub>.P channels in TG myocytes are very insensitive to inhibitory ATP. Under normal metabolic conditions, steady-state outward K<sub>sup</sub>.+ currents measured under whole cell voltage clamp were elevated in TG myocytes, consistent with threshold K<sub>sub</sub>.A<sub>sub</sub>.T<sub>sub</sub>.P activation, but neither the monophasic action potential measured in isolated hearts nor transmembrane action potential measured in right ventricular muscle preparations were shortened at physiological pacing cycles. Taken together, these results suggest that there is a compensatory remodeling of excitation-contraction coupling in TG myocytes. Whereas there were no obvious differences in other K<sub>sup</sub>.+ conductances, peak L-type Ca<sub>sup</sub>.2<sub>sup</sub>.+ current (I<sub>sub</sub>.C<sub>sub</sub>.a) density (-16.42 ± 2.37 pA/pF) in the TG was increased compared with the wild type (-8.43 ± 1.01 pA/pF). Isoproterenol approximately doubled both I<sub>sub</sub>.C<sub>sub</sub>.a and contraction amplitude in wild-type myocytes but failed to induce a significant increase in TG myocytes. Baseline and isoproterenol-stimulated cAMP concentrations were not different in wild-type and TG hearts, suggesting that the enhancement of I<sub>sub</sub>.C<sub>sub</sub>.a in the latter does not result from elevated cAMP. Collectively, the data demonstrate that a compensatory increase in I<sub>sub</sub>.C<sub>sub</sub>.a counteracts a mild activation of ATP-insensitive K<sub>sub</sub>.A<sub>sub</sub>.T<sub>sub</sub>.P channels to maintain the action potential duration and elevate the inotropic state of TG hearts.

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ACCESSION NUMBER: 2003-0244376 PASCAL

COPYRIGHT NOTICE: Copyright .COPYRG. 2003 INIST-CNRS. All rights reserved.

TITLE (IN ENGLISH): Argatroban, specific thrombin inhibitor, induced phenotype change of cultured rabbit vascular smooth muscle cells

AUTHOR: YOSHINAGA Masatomi; SUNAGAWA Masanori; SHIMADA Seiji; NAKAMURA Mariko; MURAYAMA Sadayuki; KOSUGI Tadayoshi

CORPORATE SOURCE: Department of Radiology, School of Medicine, University of the Ryukyus, Nishihara, Okinawa 903-0215, Japan; 1st Department of Physiology, School of Medicine, University of the Ryukyus, 207 Uehara,

SOURCE: Nishihara, Okinawa 903-0215, Japan  
 European journal of pharmacology, (2003), 461(1),  
 9-17, 32 refs.  
 ISSN: 0014-2999 CODEN: EJPHAZ  
 DOCUMENT TYPE: Journal  
 BIBLIOGRAPHIC LEVEL: Analytic  
 COUNTRY: Netherlands  
 LANGUAGE: English  
 AVAILABILITY: INIST-13322, 354000107504620020  
 AN 2003-0244376 PASCAL  
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 AB To investigate whether Argatroban ((2R, 4R)-4-methyl-1-[N2  
 -((RS)-3-methyl-1,2,3,4-tetrahydro-8-quinolinesulfonyl)-L-arginyl]-2-  
 piperidinecarboxylic acid hydrate, a selective thrombin inhibitor, exerts  
 a direct action on phenotype conversion of vascular smooth muscle cells,  
 cultured rabbit aortic vascular smooth muscle cells were employed. Myosin  
**heavy chain** isoforms (SM1, SM2, and SMemb) mRNA  
 expressions were evaluated by in situ hybridization and reverse  
 transcription-polymerase chain reaction (RT-PCR). After the cells were  
 incubated in serum-free medium containing argatroban (10 and 50 µg/ml)  
 and platelet-derived growth factor (PDGF)-BB (10 and 50 ng/ml) for 3 h,  
 total RNA was extracted. In situ hybridization demonstrated that myosin  
**heavy-chain** isoform mRNAs were homogenously expressed  
 in argatroban- and PDGF-BB-treated cells. RT-PCR revealed that SM1/SM2  
 mRNA expressions were not changed with argatroban, while SMemb mRNA  
 expression was increased to 1.6-fold with a statistical significance  
 (P<0.05). Treatment with argatroban (10 and 50 µg/ml) at 24 h did not  
 change SM1/SM2 mRNA expressions. Although SMemb mRNA expression was  
 slightly increased, there was no statistical significance. Other  
 phenotype markers including plasminogen activator inhibitor-1 (PAI-1) and  
 β-actin mRNAs were also significantly increased by argatroban. In  
 conclusion, argatroban can directly induce phenotype conversion of  
 vascular smooth muscle cells with the resultant up-regulation of SMemb,  
 PAI-1, and β-actin mRNAs.

L27 ANSWER 19 OF 23 PASCAL COPYRIGHT 2005 INIST-CNRS. ALL RIGHTS RESERVED.  
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ACCESSION NUMBER: 2002-0387181 PASCAL  
 COPYRIGHT NOTICE: Copyright .COPYRGT. 2002 INIST-CNRS. All rights  
 reserved.  
 TITLE (IN ENGLISH): Neuropeptide Y Y.sub.1 receptor regulates protein  
 turnover and constitutive gene expression in  
 hypertrophying cardiomyocytes  
 AUTHOR: NICHOLL Suzanne M.; BELL David; SPIERS J. Paul;  
 MCDERMOTT Barbara J.  
 CORPORATE SOURCE: Department of Therapeutics and Pharmacology, Centre  
 for Cardiovascular and Genetics Research. School of  
 Medicine. The Queen's University of Belfast, Whitla  
 Medical Building, 97 Lisburn Road, Belfast BT9 7BL,  
 Northern Ireland, United Kingdom  
 SOURCE: European journal of pharmacology, (2002), 441(1-2),  
 23-34, refs. 1 p.1/2  
 ISSN: 0014-2999 CODEN: EJPHAZ  
 DOCUMENT TYPE: Journal  
 BIBLIOGRAPHIC LEVEL: Analytic  
 COUNTRY: Netherlands  
 LANGUAGE: English  
 AVAILABILITY: INIST-13322, 354000101267540030  
 AN 2002-0387181 PASCAL  
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 AB Increased levels of neuropeptide Y correlate with severity of left  
 ventricular hypertrophy in vivo. At cardiomyocyte level, hypertrophy is  
 characterised by increased mass and altered phenotype. The aims were to

determine the contributions of increased synthesis and reduced degradation of protein to neuropeptide Y-mediated increase in mass, assess effects on gene expression, and characterise neuropeptide Y Y receptor subtype involvement. Neuropeptide Y (10 nM) increased protein mass of adult rat ventricular cardiomyocytes maintained in culture (24 h) (16%>basal and de novo protein synthesis (incorporation of [<sup>3</sup>H]-phenylalanine) (18%>basal)). Neuropeptide Y (100 nM) prevented degradation of existing protein at 8 h. Actinomycin D (5 µM) attenuated increases in protein mass to neuropeptide Y (<= 1 nM) but not to neuropeptide Y (10 nM). [Leu<sup>3</sup>, Pro<sup>3</sup>, Arg<sup>4</sup>]neuropeptide Y (10 nM), an agonist at neuropeptide Y Y<sub>1</sub> receptors, increased protein mass (25%>basal) but did not stimulate protein synthesis. Neuropeptide Y-(3-36) (10 nM), an agonist at neuropeptide Y Y<sub>2</sub> receptors, increased protein mass (29%>basal) and increased protein synthesis (13%>basal), respectively. Actinomycin D (5 µM) abolished the increase in protein mass elicited by neuropeptide Y-(3-36) but not that by [Leu<sup>3</sup>, Pro<sup>3</sup>, Arg<sup>4</sup>]neuropeptide Y. BIBP3226 [(R)-N<sup>2</sup>-(diphenylacetyl)-N-(4-hydroxyphenylmethyl)-n-arginine amide] (1 µM), a neuropeptide Y Y<sub>1</sub> receptor subtype-selective antagonist, and T<sub>4</sub> [neuropeptide Y-(33-36)]<sub>4</sub>, a neuropeptide Y Y<sub>2</sub> receptor subtype-selective antagonist, attenuated the increase in protein mass to 100 nM neuropeptide Y by 68% and 59%, respectively. Neuropeptide Y increased expression of the constitutive gene, myosin **light chain**-2 (MLC-2), maximally at 12 h (4.7-fold>basal) but did not induce (t <= 36 h) expression of foetal genes (atrial natriuretic peptide (ANP), skeletal-α-actin and myosin **heavy chain**-β). This increase was attenuated by 86% and 51%, respectively, by BIBP3226 (1 µM) and T<sub>4</sub> [neuropeptide Y-(33-36)]<sub>4</sub> (100 nM). [Leu<sup>3</sup>, Pro<sup>3</sup>, Arg<sup>4</sup>]neuropeptide Y (100 nM) (2.4-fold>basal) and peptide YY-(3-36) (100 nM) (2.3 fold>basal) increased expression of MLC-2 mRNA at 12 h. In conclusion, initiation of cardiomyocyte hypertrophy by neuropeptide Y requires activation of both neuropeptide Y Y<sub>1</sub> and neuropeptide Y Y<sub>2</sub> receptors and is associated with enhanced synthesis and attenuated degradation of protein together with increased expression of constitutive genes but not reinduction of foetal genes.

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ACCESSION NUMBER: 1997-0013845 PASCAL  
COPYRIGHT NOTICE: Copyright .COPYRGT. 1997 INIST-CNRS. All rights reserved.  
TITLE (IN ENGLISH): Hypoxia-induced pulmonary arterial contraction appears to be dependent on myosin **light chain** phosphorylation  
AUTHOR: ZHAO Y.; RHOADES R. A.; PACKER C. S.  
CORPORATE SOURCE: Department of Physiology and Biophysics, Indiana University School of Medicine, Indianapolis, Indiana 46202-5120, United States  
SOURCE: American journal of physiology. Lung cellular and molecular physiology, (1996); 15(5), L768-L774, 31 refs.  
ISSN: 1040-0605  
DOCUMENT TYPE: Journal  
BIBLIOGRAPHIC LEVEL: Analytic  
COUNTRY: United States  
LANGUAGE: English  
AVAILABILITY: INIST-22200, 354000067167410110  
AN 1997-0013845 PASCAL  
CP Copyright .COPYRGT. 1997 INIST-CNRS. All rights reserved.  
AB The signal transduction pathway of hypoxic pulmonary arterial contraction has not been elucidated. Phosphorylation of the 20-kDa myosin **light chain** (MLC.sub.2.sub.0) is thought to be

essential for vascular muscle contraction. However, there are reports that smooth muscle will contract in response to nonphysiological stimuli such as phorbol esters without the involvement of MLC.sub.2.sub.0 phosphorylation. The purpose of this study was to determine if hypoxia-induced pulmonary arterial contraction is dependent on MLC.sub.2.sub.0 phosphorylation. Isolated rat pulmonary and carotid (for comparative purposes) arterial strips were contracted with 80 mM KCl to establish maximum active tension in response to membrane depolarization. The strips were then stimulated with one of the following: 30 mM KCl, 1  $\mu$ M phenylephrine, 0.01  $\mu$ M angiotensin II, 1  $\mu$ M phorbol 12-myristate 13-acetate (PMA), or hypoxia (95% N.sub.2-5% CO.sub.2). In some experiments ML-9, a myosin **light chain** kinase inhibitor, or calphostin C, a protein kinase C (PKC) inhibitor, was introduced into the bath before hypoxia. Isometric tension was recorded as a function of time. Muscle strips were freeze-clamped (liquid N<sub>2</sub>) at various time points during the course of responses to the various stimuli. MLC.sub.2.sub.0 phosphorylation levels were measured by urea-glycerol gel electrophoresis followed by Western blot procedure. Results show that increased MLC.sub.2.sub.0 phosphorylation correlates with initiation of pulmonary arterial smooth muscle contraction in response to all agonists with the exception of PMA, a known activator of PKC. The MLC.sub.2.sub.0 phosphorylation levels correlate with tension development in response to hypoxia, and ML-9 abolished the hypoxic contractions. In contrast, hypoxia relaxed carotid arterial muscle, and there was a corresponding decrease in the MLC.sub.2.sub.0 phosphorylation level. In conclusion, hypoxia appears to result in MLC.sub.2.sub.0 phosphorylation-mediated contraction in conduit pulmonary arterial muscle and in MLC.sub.2.sub.0 dephosphorylation-mediated relaxation in systemic arterial muscle.

L27 ANSWER 21 OF 23 PASCAL COPYRIGHT 2005 INIST-CNRS. ALL RIGHTS RESERVED.  
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ACCESSION NUMBER: 1996-0474477 PASCAL  
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TITLE (IN ENGLISH): Purification and sequencing of yellow mustard seed napin small and large chains that are phosphorylated by plant calcium-dependent protein kinases and are calmodulin antagonists  
AUTHOR: NEUMANN G. M.; CONDRON R.; POLYA G. M.  
CORPORATE SOURCE: School of Biochemistry, La Trobe University, Bundoora, Victoria 3083, Australia  
SOURCE: Plant science : (Limerick), (1996), 119(1-2), 49-66, 44 refs.  
ISSN: 0168-9452 CODEN: PLSCE4  
DOCUMENT TYPE: Journal  
BIBLIOGRAPHIC LEVEL: Analytic  
COUNTRY: Ireland  
LANGUAGE: English  
AVAILABILITY: INIST-15982, 354000066320390060

AN 1996-0474477 PASCAL

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AB A multiplicity of small (S) and large (L) napin subunits were purified from yellow mustard (*Sinapis alba* L.) seeds by a protocol involving extraction, successive batch-wise cation exchange chromatography on carboxymethylcellulose (CM52), cation exchange HPLC on an SP5PW column and reversed phase HPLC on a C18 column. Initial cation exchange HPLC resolved 4 major zones of proteins (M1, M2, N1 and N2) that can be phosphorylated by plant Ca.sup.2.sup.-dependent protein kinase (CDPK). Electrospray ionization mass spectrometry (ESMS) revealed that M1 and M2 are 6 kDa proteins, later identified as  $\gamma$ -thionin-related proteins. ESMS of fractions N1 and N2 revealed the presence of 14.5 kDa proteins identified as napin complexes, each composed of a

single small subunit linked to a single large subunit and involving 4 disulphide linkages. The napin complexes (N1A, N1B, N1C, N1D, N2A, N2B and N2C) were disrupted and the constituent small subunits (S1, S2 and S3) and large subunits (L1A, L1B, L1C, L2A, L2B and L2C) were resolved by reversed phase HPLC and precise average molecular masses determined by ESMS. The small and large subunits have average molecular masses of about 4.4 kDa and 10.1 kDa, respectively. The masses of each napin complex can be precisely accounted for from the masses of the constituent subunits. Thus the major complex N2A ( $14\,569 \pm 3$  Da) is evidently composed of S3 ( $4434.0 \pm 1.5$  Da) and L2A ( $10142.5 \pm 1.5$  Da) and involves 4 disulphides (loss of 8.0 Da), the expected mass of S3 + L2A-8H being  $14\,569 \pm 2$  Da. The yellow mustard napin large chain L2A is phosphorylated by wheat CDPK on Ser.sup.6.sup.0 within the sequence LQHVIS.sup.6.sup.0RIY. The complete sequence of this and other large (and small) napin subunits were determined from Edman sequencing and/or ESMS data by comparison with published napin sequences. Yellow mustard seed CM52-binding fractions decrease the Ca.sup.2.sup.+ dependent fluorescence emission of dansyl-CaM and yellow mustard small and large chains inhibit CaM-dependent myosin **light chain** kinase (MLCK).

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ACCESSION NUMBER: 1996-0429472 PASCAL

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TITLE (IN ENGLISH): Molecular analysis of the leukaemic B cell in adult and childhood acute lymphoblastic leukaemia

AUTHOR: COYLE L. A.; PAPAIOANNOU M.; YAXLEY J. C.; CHIM J. S.; ATTARD M.; HOFFBRAND A. V.; FORONI L.

CORPORATE SOURCE: Department of Haematology, Royal Free Hospital School of Medicine, London, United Kingdom

SOURCE: British journal of haematology, (1996), 94(4), 685-693, refs. 1 p.1/4

ISSN: 0007-1048 CODEN: BJHEAL

DOCUMENT TYPE: Journal; (research paper)

BIBLIOGRAPHIC LEVEL: Analytic

COUNTRY: United Kingdom

LANGUAGE: English

AVAILABILITY: INIST-7597, 354000066065950160

AN 1996-0429472 PASCAL

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AB Immunoglobulin **heavy chain** gene (IgH gene)

rearrangements are found in the majority of cases of B-lineage acute lymphoblastic leukaemia (ALL). We have examined bone marrow samples taken at presentation or relapse from 109 patients (79 adults and 30 children) and have performed sequence analysis of the complementarity determining region 3 (CDR3) on 65 alleles from 54 patients. We aimed to define immunoglobulin **heavy chain** (IgH) variable segment family use and investigate biological and structural features of the B cell in adult and childhood ALL. Using the FR1 fingerprinting method. a rearranged band was identified in 70 (89%) of 79 adult ALL and in 29 (97%) of 30 childhood ALL. This study found no preferential use or selection of IgH **VH** genes and no statistically significant structural differences between normal and leukaemic B cells in either adult and childhood ALL. An equal proportion of amplifiable cases of adult and childhood ALL uses more than one **VH** family gene (24/70, 34%, and 8/29, 27.5%, respectively). There were no significant differences in the structure or size of the CDR3 region and the variable (V) or joining (J) segment use in ALL patients compared to normal B cells. We observed that the **N2** region was shorter than N 1 in children whereas the opposite was observed in adults (not statistically significant). The J4 segment was a more common rearrangement in children than in adults, and in both groups J4 was more frequently associated with

multiple D segment VDJ rearrangements. An increase in VH6 use in leukaemic alleles compared to normal 13 lymphocytes (2%) was observed but it was not statistically significant in our group of patients. Amongst children and adults, in-frame CDR3 junctions occurred in 78% and 64% of rearranged alleles, respectively, compared to 75% of inframe sequences reported by others to occur among normal B cells.

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ACCESSION NUMBER: 1990-0085388 PASCAL  
TITLE (IN ENGLISH): Detection of human and marmoset immunoglobulin  
**heavy chain** by a polyclonal  
antiserum to a marmoset immunoglobulin-related T cell  
product

AUTHOR: SPEIDEL M. T.; MARCHALONIS J. J.

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SOURCE: Experimental and clinical immunogenetics, (1989),  
6(4), 245-250, 15 refs.

ISSN: 0254-9670 CODEN: ECIME4

DOCUMENT TYPE: Journal

BIBLIOGRAPHIC LEVEL: Analytic

COUNTRY: Switzerland

LANGUAGE: English

AVAILABILITY: CNRS-20714

AN 1990-0085388 PASCAL

AB We show that a polyclonal rabbit antiserum raised against a purified  
monoclonal T cell component from a lower primate (cotton-topped marmoset)  
reacts by immunoblot transfer (Western Blot analysis) with serum  
immunoglobulin of man and marmoset. The antigenic component had an  
approximate mass of 68 kilodaltons and was isolated by immune-affinity  
chromatography from culture fluid in which the marmoset T cell leukemia  
70-N2 had been grown. The reaction with human serum  
immunoglobulin is with a subset of the IgG molecules and is localized to  
the  $\gamma$  **heavy chain**